

FILE 'MEDLINE, EMBASE, BIOSIS' ENTERED AT 10:52:48 ON 07 MAY 2004

L1 20671 S HYPERTENSION AND ANIMAL MODEL  
L2 1079 S L1 AND ESSENTIAL HYPERTENSION  
L3 2 S L2 AND VEGF

=> s l2 and accepted

L4 5 L2 AND ACCEPTED

=> duplicate remove l4

DUPLICATE PREFERENCE IS 'EMBASE, BIOSIS'

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L5 5 DUPLICATE REMOVE L4 (0 DUPLICATES REMOVED)

=> d 1-5

L5 ANSWER 1 OF 5 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN  
AN 2002:42259 BIOSIS  
DN PREV200200042259  
TI Intrauterine programming of nephron number: The fetal flaw revisited.  
AU Marchand, Michael C.; Langley-Evans, Simon C. [Reprint author]  
CS Division of Nutritional Biochemistry, University of Nottingham, Sutton  
Bonington Campus, Loughborough, LE12 5RD, UK  
Simon.Langley-Evans@nottingham.ac.uk  
SO JN Journal of Nephrology, (September-October, 2001) Vol. 14, No. 5, pp.  
327-331. print.  
ISSN: 1121-8428.  
DT Article  
General Review; (Literature Review)  
LA English  
ED Entered STN: 2 Jan 2002  
Last Updated on STN: 25 Feb 2002

L5 ANSWER 2 OF 5 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.  
on STN  
AN 97238915 EMBASE  
DN 1997238915  
TI Diet, genetics and **hypertension**.  
AU Preuss H.G.  
CS Dr. H.G. Preuss, Georgetown University Medical Center, 4000 Reservoir Rd  
NW, Washington, DC 20007, United States  
SO Journal of the American College of Nutrition, (1997) 16/4 (296-305).  
Refs: 164  
ISSN: 0731-5724 CODEN: JONUDL  
CY United States  
DT Journal; General Review  
FS 005 General Pathology and Pathological Anatomy  
006 Internal Medicine  
017 Public Health, Social Medicine and Epidemiology  
LA English  
SL English

L5 ANSWER 3 OF 5 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.  
on STN  
AN 95362379 EMBASE  
DN 1995362379  
TI Transgenic animals in the study of blood pressure regulation and  
**hypertension**.  
AU Thompson M.W.; Merrill D.C.; Yang G.; Robillard J.E.; Sigmund C.D.  
CS Transgenic Animal Facility, Dept. of Medicine, Univ. of Iowa College of  
Medicine, Iowa City, IA 52242, United States  
SO American Journal of Physiology - Endocrinology and Metabolism, (1995)  
269/5 32-5 (E793-E803).

ISSN: 0193-1849 CODEN: AJPM

CY United States  
 DT Journal; General Review  
 FS 002 Physiology  
 006 Internal Medicine  
 018 Cardiovascular Diseases and Cardiovascular Surgery  
 022 Human Genetics  
 037 Drug Literature Index  
 LA English  
 SL English

L5 ANSWER 4 OF 5 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN  
 AN 1992:162824 BIOSIS  
 DN PREV199293085149; BA93:85149  
 TI SPONTANEOUSLY HYPERTENSIVE AND WISTAR KYOTO RATS ARE GENETICALLY  
 DISPARATE.  
 AU H'DOUBLER P B JR [Reprint author]; PETERSEN M; SHEK W; AUCHINCLOSS H;  
 ABBOTT W M; ORKIN R W  
 CS VASCULAR DIV, SURG SERV, MASSACHUSETTS GENERAL HOSP, DEP SURG, HARVARD MED  
 SCH, BOSTON, MASS 02114, USA  
 SO Laboratory Animal Science, (1991) Vol. 41, No. 5, pp. 471-473.  
 CODEN: LBASAE. ISSN: 0023-6764.  
 DT Article  
 FS BA  
 LA ENGLISH  
 ED Entered STN: 31 Mar 1992  
 Last Updated on STN: 31 Mar 1992

L5 ANSWER 5 OF 5 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.  
 on STN  
 AN 85033735 EMBASE  
 DN 1985033735  
 TI Studies on the effect of a chemical sympathectomy on the enzyme pattern in  
 the heart of spontaneously hypertensive rats (SHR) fed diets supplemented  
 with different polyunsaturated fatty acids (PUFA).  
 AU Papias B.; Wagenknecht C.; Konig M.-L.; et al.  
 CS Institute of Pathological and Clinical Biochemistry, Humboldt-University,  
 Berlin, Germany  
 SO Biomedica Biochimica Acta, (1984) 43/8-9 (S179-S183).  
 CODEN: BBIADT  
 CY Germany  
 DT Journal  
 FS 003 Endocrinology  
 018 Cardiovascular Diseases and Cardiovascular Surgery  
 029 Clinical Biochemistry  
 002 Physiology  
 LA English

=> d 1-5 abs

L5 ANSWER 1 OF 5 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN  
 AB A broad range of epidemiological evidence supports the hypothesis that  
 risk of **essential hypertension**, coronary heart disease  
 and non-insulin dependent diabetes is, in part, determined before birth.  
 This phenomenon, termed programming, is now the subject of intensive  
 investigation in order to determine possible underlying mechanisms. It is  
 widely **accepted** that maternal nutritional status in pregnancy is  
 a major programming influence upon the fetus. This review considers the  
 hypothesis that nephron number in humans is determined by prenatal  
 nutrition. An increasing number of human studies indicate that the  
 developing kidney is particularly vulnerable to the adverse effects of  
 fetal growth retarding influences. In animals, growth retarding diets or  
 other insults which have an impact upon the development of cardiovascular

functions, also appear to impact upon nephron number. However, it is possible that **hypertension** and reduced renal reserve merely coincide and are not causally associated.

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AB It is generally **accepted** that genetics play a significant role in the pathogenesis of **hypertension**. Since **hypertension** often follows kidney transplantation, candidate genes have been sought and found in the kidneys of rats and humans. One well-recognized, inherited influence on blood pressure (Bp) occurs via abnormal renal sodium handling in vivo. Further, abnormal renal sodium handling is seen in isolated kidneys of genetically hypertensive rats. People who have a relative inability to handle a sodium load properly, and retain it inappropriately, often develop high BP and are referred to as 'salt-sensitive'. More than half of patients diagnosed with **essential hypertension** are salt-sensitive. In contrast to the deleterious effects associated with high sodium intake, many believe that ingestion of more potassium, calcium, and magnesium may influence BP favorably. The beneficial effects of these ions work, at least in part, through an effect on sodium balance, i.e., a diuretic influence. In support of this concept, they lower BP more effectively in salt-sensitive hypertensives. Refined carbohydrates and saturated fats are also associated with salt retention and **hypertension**. Thus, dietary factors, working directly on their own and/or indirectly via effects on genetic mechanisms, may alter BP favorably or unfavorably.

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AB It is generally **accepted** that the etiology of **essential hypertension** is due to a complex interplay of genetic and environmental factors. A great deal of research effort over the past ten years has been focused on the identification of genes the variants of which predispose individuals to high blood pressure. Consequently, transgenic and knockout animals have become important research tools, providing experimental systems in which defined genetic manipulations can be introduced on uniform genetic backgrounds while minimizing environmental variation. These animal models have provided the means by which candidate genes thought to be involved in blood pressure regulation have been studied. Furthermore, these models can be used to test the significance of genes and gene variants identified via genome-wide searches as potential causes of **hypertension**. The purpose of this review is to provide a brief discussion of transgenic and knockout methodology and its application to study the genetic basis of **hypertension**.

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AB Spontaneously hypertensive rats (SHR) are one of the most common animal models used to study **essential hypertension** in humans. Because SHR and normotensive Wistar Kyoto (WKY) rats were both established from the same parenteral, normotensive Wistar stock, WKY animals have been used almost exclusively as control animals in studies of SHR. Recently, the suitability of WKY rats as normotensive controls for SHR has been challenged. To establish whether or not SHR and WKY rats share the same immunologic backgrounds, we initially performed a series of skin grafting experiments on these animals. In all cases, grafts of SHR donor skin to WKY recipients and of WKY donor skin to SHR recipients resulted in complete rejection within 7 to 10 days. In addition, grafts of WKY donor skin to other WKY recipients resulted in graft rejection. By contrast, skin grafts between SHRs were always **accepted**. To further characterize the genetic distinctions between SHR and WKY rats, allelic profiles based on a series of immunologic and biochemical markers were established for each strain. These findings clearly establish that SHR and WKY rats differ at the major histocompatibility complex, in specific

blood group antigens, and in a panel of isozymic markers. Moreover, whereas SHRs have the same genetic profiles irrespective of source, some colonies of WKY rats are outbred, as judged by their variant allelic profiles.

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AB Spontaneously hypertensive rats are generally **accepted** as a suitable model for **essential hypertension** in man at least with regard to some etiological and pathogenetic aspects. In these animals the genetically determined **hypertension** manifests itself at an age of about 8-10 weeks. The cause of blood pressure elevation is hitherto not fully clear. It is suggested that an increased activity of the sympathetic nervous system which has been described in early stages of spontaneous **hypertension** in rats serves as an initiating mechanism for **hypertension**. This elevated sympathetic activity might at least be partly related to disturbances in the metabolism of PUFA and prostaglandins taking into account the known negative feedback of E prostaglandins on transmitter release (1). The influence of LA supplemented diet on catecholamines and dopamine  $\beta$ -hydroxylase in the adrenals of SHR (2) as well as the blood pressure lowering effect of prenatal feeding PUFA rich diets (3) are in agreement with this suggestion. In a previous study we have shown that not only the blood pressure of SHR but also **hypertension**-linked complications reflected by alterations of the enzyme pattern in heart and liver can be beneficially influenced by prenatal application of PUFA enriched diets. It was the purpose of the present study to investigate: 1. the influence of postnatal feeding PUFA rich and PUFA deficient diets on blood pressure and myocardial enzyme pattern, 2. the effectiveness of different PUFA in influencing blood pressure and enzyme pattern, 3. the effect of chemical sympathectomy in PUFA rich and PUFA deficient SHR.

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